? b 155
22may03 08:57:46 User208669 Session D2300.1
$0.29 0.084 DialUnits File1
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$0.29 Estimated cost this search
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File 155:MEDLINE(R) 1966-2003/May W3
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Set Items Description
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? s hvea and py<1999
28 HVEA
10110473 PY<1999
S1 4 HVEA AND PY<1999
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DIALOG(R)File 155:MEDLINE(R)
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11438266 98321161 PMID: 9657005
A cell surface protein with herpesvirus entry activity (HveB) confers susceptibility to infection by mutants of herpes simplex virus type 1, herpes simplex virus type 2, and pseudorabies virus.
Warner M S; Geraghty R J; Martinez W M; Montgomery R I; Whitbeck J C; Xu R; Eisenberg R J; Cohen G H; Spear P G
Department of Microbiology-Immunology, Northwestern University Medical School, Chicago, Illinois 60611, USA.
Virology (UNITED STATES) Jun 20 1998, 246 (1) p179-89, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI36293; AI; NIAID; NS30606; NS; NINDS; NS36731; NS; NINDS; +
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Certain mutant strains of herpes simplex virus type 1 (HSV-1) are unable to infect cells in which entry is dependent on HVEM, the previously described herpesvirus entry mediator designated here as herpesvirus entry protein A (HveA). These mutant viruses can infect other cells where entry is apparently dependent on other co-receptors. The mutant virus HSV-1(KOS)Rid1 was used to screen a human cDNA expression library for ability of transfected plasmids to convert resistant Chinese hamster ovary cells to susceptibility to virus entry. A plasmid expressing the previously described poliovirus receptor-related protein 2 (Prr2) was isolated on the basis of this activity. This protein, designated here as HveB, was shown to mediate the entry of three mutant HSV-1 strains that cannot use HVEM as co-receptor, but not wild-type HSV-1 strains. HveB also mediated the entry of HSV-2 and pseudorabies virus but not bovine herpesvirus type 1. HveB was expressed in some human neuronal cell lines, fibroblastic cells, keratinocytes, and primary activated T lymphocytes. Antibodies specific for HveB blocked infection of HveB-expressing CHO cells and a human fibroblastic cell strain HEL299. Differences in ability of HSV-1 and HSV-2 strains to use HveB for entry should influence the types of cells that can be infected and thereby account in part for serotype and strain differences in tissue tropism and pathogenicity.
Record Date Created: 19980727
Record Date Completed: 19980727

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DIALOG(R)File 155:MEDLINE(R)
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11404081 98285738 PMID: 9621040
HveA (herpesvirus entry mediator A), a coreceptor for herpes simplex virus entry, also participates in virus-induced cell fusion.
Terry-Allison T; Montgomery R I; Whitbeck J C; Xu R; Cohen G H; Eisenberg R J; Spear P G
Department of Microbiology-Immunology, Northwestern University Medical School, Chicago, Illinois 60611, USA.
Journal of virology (UNITED STATES) Jul 1998, 72 (7) p5802-10, ISSN 0022-538X Journal Code: 0113724
Contract/Grant No.: PO1 NS30606; NS; NINDS; R01 CA21776; CA; NCI; RO1 NS36731; NS; NINDS; +
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The purpose of this study was to determine whether a cell surface protein that can serve as coreceptor for herpes simplex virus type 1 (HSV-1) entry, herpesvirus entry mediator (previously designated HVEM but renamed HveA), also mediates HSV-1-induced cell-cell fusion. We found that transfection of DNA from KOS-804, a previously described HSV-1 syncytial (Syn) strain whose Syn mutation was mapped to an amino acid substitution in gK, induced numerous large syncytia on HveA-expressing Chinese hamster ovary cells (CHO-HVEM12) but not on control cells (CHO-C8). Antibodies specific for gD as well as for HveA were effective inhibitors of KOS-804-induced fusion, consistent with previously described direct interactions between gD and HveA. Since mutations in gD determine the ability of HSV-1 to utilize HveA for entry, we examined whether the form of virally expressed gD also influenced the ability of HveA to mediate fusion. We produced a recombinant virus carrying the KOS-804 Syn mutation and the KOS-Rid1 gD mutation, which significantly reduces viral entry via HveA, and designated it KOS-SR1.

KOS-SR1 DNA had a markedly reduced ability to induce syncytia on CHO-HVEM12 cells and a somewhat enhanced ability to induce syncytia on CHO-C8 cells. These results support previous findings concerning the relative abilities of KOS and KOS-Rid1 to infect CHO-HVEM12 and CHO-C8 cells. Thus, HveA mediates cell-cell fusion as well as viral entry and both activities of HveA are contingent upon the form of gD expressed by the virus.

Record Date Created: 19980701

Record Date Completed: 19980701

? log hold

22may03 09:03:15 User208669 Session D2300.2

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$0.42 2 Type(s) in Format 7

$0.42 6 Types

$2.77 Estimated cost File155

$1.40 TELNET

$4.17 Estimated cost this search

$4.46 Estimated total session cost 0.819 DialUnits

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